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Case Report

IgA Nephropathy Presenting With Recurrent Hematuria Following Upper Respiratory Infection: A Case Report

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Abstract

Background: IgA nephropathy is the most common primary glomerulonephritis worldwide and is characterized by mesangial deposition of immunoglobulin A. The disease demonstrates a broad spectrum of clinical manifestations ranging from asymptomatic microscopic hematuria to progressive renal dysfunction. Early recognition remains crucial because timely intervention can significantly slow disease progression.
Case Presentation: We report the case of a 28-year-old male who presented with intermittent episodes of dark-colored urine occurring shortly after upper respiratory tract infections. The patient also reported mild pedal edema. Laboratory investigations revealed persistent microscopic hematuria and proteinuria with mildly elevated serum creatinine. Complement levels were within normal limits and autoimmune markers were negative. Ultrasonography demonstrated normal-sized kidneys with preserved corticomedullary differentiation. Renal biopsy revealed mesangial proliferation on light microscopy, while immunofluorescence demonstrated dominant mesangial deposition of immunoglobulin A with co-deposition of complement component C3. These findings confirmed the diagnosis of IgA nephropathy. The patient was managed with renin–angiotensin system blockade and lifestyle modification, resulting in improvement in proteinuria and stabilization of renal function during follow-up.
Conclusion: This case highlights the importance of considering IgA nephropathy in patients presenting with recurrent hematuria following mucosal infections. Early diagnosis and appropriate management are essential to prevent long-term renal complications.

Keywords: *IgA nephropathy, Berger disease, hematuria, glomerulonephritis, renal biopsy, proteinuria, syntharyngitic hematuria, chronic kidney disease*

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Introduction

IgA nephropathy, also known as Berger disease, is the most common primary glomerulonephritis worldwide and is characterized by the deposition of immunoglobulin A in the mesangium of the glomeruli.[1] The disease demonstrates a wide spectrum of clinical manifestations, ranging from asymptomatic microscopic hematuria detected on routine evaluation to progressive renal dysfunction leading to chronic kidney disease.[2] Classically, patients present with recurrent episodes of macroscopic hematuria occurring shortly after upper respiratory tract infections, a phenomenon often described as synpharyngitic hematuria.[3]
Despite its well-recognized pathophysiology, the clinical presentation of IgA nephropathy can be highly variable and occasionally atypical, which may delay diagnosis and appropriate management.[4] Some patients may present with isolated proteinuria, hypertension, or nonspecific urinary abnormalities without the classical history of infection-associated hematuria, posing a diagnostic challenge in routine clinical practice.
Given that early identification and timely initiation of supportive therapy are crucial in slowing disease progression, recognizing subtle or atypical presentations becomes clinically important. Here, we report a case of IgA nephropathy presenting with recurrent hematuria temporally associated with upper respiratory tract infections, highlighting key diagnostic considerations and reinforcing the importance of early clinical suspicion in preventing long-term renal complications.

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Case Presentation

A 28-year-old male presented to the outpatient department with complaints of intermittent episodes of dark-colored urine for the past three months. The patient reported that the discoloration of urine was particularly noticeable following episodes of upper respiratory tract infection associated with sore throat and low-grade fever. He also reported mild bilateral pedal edema for the preceding two weeks. There was no history of dysuria, flank pain, decreased urine output, or passage of renal calculi. The patient denied any history of diabetes mellitus, systemic hypertension, autoimmune disorders, or chronic medication use. Family history of kidney disease was absent.

On physical examination, the patient was hemodynamically stable. His blood pressure was 146/92 mmHg, pulse rate was 84 beats per minute, and temperature was within normal limits. Mild bilateral pitting pedal edema was noted. Systemic examination of cardiovascular, respiratory, and abdominal systems was otherwise unremarkable.

Initial laboratory investigations revealed a hemoglobin level of 13.8 g/dL, total leukocyte count of 8,700/mm³, and platelet count of 240,000/mm³. Serum biochemistry demonstrated a serum creatinine level of 1.6 mg/dL and blood urea nitrogen of 28 mg/dL. Serum electrolytes were within normal limits. Liver function tests were unremarkable. Serum albumin was 3.4 g/dL, and lipid profile showed mildly elevated total cholesterol.

Urinalysis revealed significant microscopic hematuria with more than 100 red blood cells per high-power field and proteinuria (2+). Twenty-four-hour urinary protein excretion was measured at 1.2 g/day. Urine culture was sterile. Complement levels (C3 and C4) were within normal limits, and serological investigations including antinuclear antibody (ANA), anti–double stranded DNA antibodies, and antineutrophil cytoplasmic antibodies (ANCA) were negative. These findings helped exclude secondary causes of glomerulonephritis.

Ultrasonography of the abdomen revealed normal-sized kidneys with preserved corticomedullary differentiation and no evidence of hydronephrosis or renal calculi. In view of persistent hematuria and proteinuria with mildly impaired renal function, a percutaneous renal biopsy was performed for definitive diagnosis.

Light microscopic examination of the renal biopsy specimen revealed mesangial hypercellularity with expansion of the mesangial matrix involving multiple glomeruli. Some glomeruli showed segmental sclerosis. No significant endocapillary proliferation or crescent formation was observed. The tubulointerstitial compartment showed mild interstitial fibrosis with focal tubular atrophy.

Immunofluorescence microscopy demonstrated dominant mesangial deposition of IgA with co-deposition of complement component C3 in a granular pattern. Electron microscopy revealed electron-dense deposits predominantly located in the mesangial regions. These histopathological findings were consistent with a diagnosis of IgA nephropathy.

Based on the clinical, laboratory, and histopathological findings, the patient was diagnosed with primary IgA nephropathy. The patient was initiated on supportive therapy including renin-angiotensin system blockade with an angiotensin-converting enzyme inhibitor for blood pressure control and reduction of proteinuria. Lifestyle modifications including salt restriction and regular follow-up were advised.

During follow-up at three months, the patient demonstrated improvement in blood pressure control and a reduction in proteinuria to 0.6 g/day, with stable renal function.

Table 1. Investigations

Parameter	Value	Reference Range
Hemoglobin	13.8 g/dL	13–17 g/dL
TLC	8700/mm³	4000–11000/mm³
Platelets	240,000/mm³	150,000–400,000/mm³
Serum Creatinine	1.6 mg/dL	0.6–1.2 mg/dL
BUN	28 mg/dL	7–20 mg/dL
Proteinuria (24h)	1.2 g/day	<150 mg/day
Urine RBCs	>100/HPF	<5/HPF
C3/C4	Normal	—
ANA, ANCA	Negative	—

Discussion

IgA nephropathy (IgAN) is the most common primary glomerulonephritis worldwide and represents a significant cause of chronic kidney disease in young adults. First described by Jean Berger and Nicole Hinglais in 1968, the condition is characterized by the deposition of IgA-containing immune complexes within the mesangium of the glomeruli.[5] The disease demonstrates considerable heterogeneity in its clinical presentation, histopathological findings, and long-term outcomes, which makes early recognition and appropriate management particularly important.[3,6]

The pathogenesis of IgA nephropathy is multifactorial and involves a complex interplay between genetic susceptibility, environmental triggers, and immune dysregulation.[7] Current evidence supports a “multi-hit” pathogenic model in which aberrantly glycosylated IgA1 molecules are produced by mucosal immune cells.[5,8] These galactose-deficient IgA1 molecules subsequently stimulate the formation of autoantibodies, leading to circulating immune complexes that deposit in the glomerular mesangium.[7,8] This deposition activates mesangial cells, induces complement activation, and triggers inflammatory pathways that ultimately result in glomerular injury and progressive renal dysfunction.

Clinically, IgA nephropathy most commonly presents with episodic macroscopic hematuria following mucosal infections such as upper respiratory tract infections.[9] However, a substantial proportion of patients present with asymptomatic microscopic hematuria, isolated proteinuria, or incidentally detected urinary abnormalities during routine health screening. In some cases, patients may develop nephritic syndrome, hypertension, or progressive renal insufficiency. The variability in clinical manifestations often complicates early diagnosis and may delay appropriate therapeutic intervention.

In the present case, the patient demonstrated recurrent episodes of hematuria temporally associated with upper respiratory tract infections, a pattern frequently described as synpharyngitic hematuria and considered characteristic of IgA nephropathy. Laboratory findings revealed persistent hematuria with moderate proteinuria and mildly impaired renal function. Serological investigations excluded secondary causes of glomerulonephritis, including autoimmune and systemic inflammatory disorders. Renal biopsy confirmed the diagnosis by demonstrating mesangial proliferation and dominant IgA deposition on immunofluorescence, which remains the gold standard for diagnosis.

Histopathological evaluation plays a crucial role in determining disease severity and prognosis.[10] The Oxford classification of IgA nephropathy has been widely adopted to standardize pathological assessment and includes evaluation of mesangial hypercellularity, endocapillary proliferation, segmental glomerulosclerosis, tubular atrophy, and interstitial fibrosis.[11] These histological parameters have been shown to correlate with long-term renal outcomes and are useful in guiding therapeutic decisions.

Management strategies for IgA nephropathy focus primarily on slowing disease progression and preventing the development of end-stage renal disease.[12] Supportive therapy, particularly strict blood pressure control and reduction of proteinuria using renin–angiotensin system blockade, forms the cornerstone of treatment. Patients with persistent proteinuria despite optimized supportive therapy may benefit from immunosuppressive treatment, most commonly corticosteroids. Recent research has also explored emerging therapies targeting specific components of the disease pathway, including complement inhibition and modulation of mucosal immune responses.

The prognosis of IgA nephropathy is variable.[13] While some patients experience a relatively benign course with stable renal function for many years, approximately 20–40% of patients may progress to end-stage renal disease over two to three decades. Risk factors associated with poor prognosis include sustained proteinuria, hypertension, impaired renal function at presentation, and significant chronic changes on renal biopsy.

This case highlights the importance of recognizing recurrent hematuria associated with mucosal infections as a potential early indicator of IgA nephropathy. Early diagnosis through timely renal biopsy and initiation of appropriate supportive therapy may help reduce disease progression and improve long-term renal outcomes. Furthermore, documenting such cases contributes to the growing body of clinical literature aimed at improving understanding of the diverse presentations and clinical course of this common glomerular disease.

In conclusion, IgA nephropathy remains the most common primary glomerulonephritis worldwide and continues to present a broad spectrum of clinical manifestations ranging from asymptomatic microscopic hematuria to progressive renal impairment. The present case highlights the importance of recognizing recurrent hematuria following mucosal infections as a potential early clinical indicator of IgA nephropathy. Early identification through appropriate clinical evaluation and renal biopsy allows timely initiation of supportive therapy aimed at reducing proteinuria and slowing disease progression. Increased clinical awareness of the variable presentation of IgA nephropathy is essential for prompt diagnosis and improved long-term renal outcomes.

Declarations

Ethics Approval: Ethical approval was not required for this case report as per institutional policy, as the study describes a single clinical case with patient consent and does not involve experimental intervention.

Patient Consent: Written informed consent was obtained from the patient for publication of the clinical details and relevant investigations included in this case report. The patient’s identity has been protected, and no identifiable personal information has been disclosed.

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